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Ringer

John Tweedy Esq with Sydney Ringer's kind regards

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Original Communications.

NOTES OF A POST-MORTEM EXAMINATION ON A CASE OF ATHETOSIS.

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In the *Practitioner* for August, 1877, I published the notes of a case of athetosis. The patient, when twenty-eight years of age, was struck down with right-sided hemiplegia, hemianæsthesia, and unilateral sweating. He subsequently recovered the lost power and sensation on the right side, but the unilateral sweating persisted. As he regained his lost power the athetosis developed itself. The patient was re-admitted May 29, 1878. He now suffered severely from his heart; he had orthopnœa; his jugulars were full, and his lips and ears livid, his legs were greatly swollen, and his expectoration bloody. His athetosis was much the same as when he left the hospital about a year before. He died June 12. I shall first give a brief account of the examination of his heart and lungs, and then a fuller account of the state of his brain.

The right auricle and ventricle were very greatly enlarged, and the auricle much hypertrophied, its walls being twice their natural thickness. The tricuspid valves were diseased and partially united at their edges, so as to cause decided tricuspid obstruction. The pulmonary artery contained an old laminated clot occupying a large part of the lumen of the vessel. The left auricle contained a small old clot in the appendix. The mitral valve was much constricted, and would only admit the tip of the forefinger, and the edge of the valve-ring was calcareous. The left ventricle was very slightly hypertrophied. The aortic valves were not quite competent, and showed slight calcareous change. The heart weighed twelve ounces. In both lungs we found pulmonary apoplexies. The walls of the pulmonary artery had undergone considerable atheromatous and calcareous degeneration.

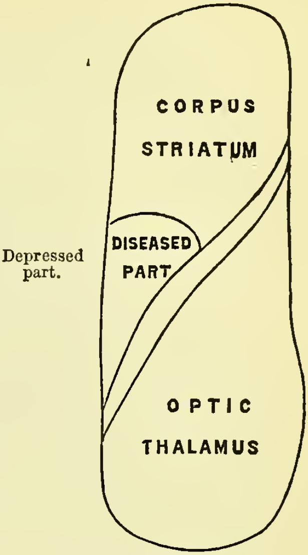
Brain.—We could not discover any trace of an occluded vessel.

The pia mater was slightly opaque and cloudy, perhaps rather more on the left than the right side, and especially so anteriorly.

The left hemisphere did not seem at all wasted. There was, perhaps, a little wasting over a very small extent, an inch in diameter, and an inch outside the longitudinal fissure and at the junction of the anterior and middle third. On stripping off the pia mater the convolution of the left side seemed to fall apart a little more than those of the right.

We then made transverse horizontal sections across the brain, and found the brain substance, judging by the naked eye, healthy till we laid bare the lateral ventricles. The parts in the right lateral ventricle looked quite healthy. In the left lateral ventricle there were evident signs of disease affecting the posterior part of the corpus striatum, that portion close to the tænia semicircularis, and which lies just outside the anterior portion of the thalamus. This part was depressed and rather hollowed out, and the membrane covering it was thickened. The anterior part of the corpus striatum and the tænia semicircularis looked healthy. The left optic thalamus was decidedly smaller and flatter than the right.

We then exposed the third ventricle and found the anterior and posterior commissures very distinct, but the soft commissure was gone. The optic thalami were now better exposed, and the diminution of the left was now more apparent, it being perhaps a quarter less than the right, the transverse measurement of the right thalamus being two and a half centimetres, that of the left two centimetres. We then made transverse vertical sections through both corpora striata. The first section was made through the anterior part of the corpora. We found the brain substance beneath the depressed portion of the left corpus much softened, the softening extending a little to the right and left of the depression. The softened tissue was a light yellow colour.



We then made another transverse section a quarter of an inch posterior to the first, and this exposed, on the left side, the anterior part of a cyst situated in the grey matter of the lenticular nucleus, just external to the white matter dividing the caudate from the lenticular nucleus. Further sections showed that this cyst had its long axis in the antero-posterior measurement of the brain and involved the lenticular ganglion, the anterior part of the cyst being mainly, if not exclusively, in the lenticular ganglion, but it extended behind the corpus striatum, lying outside and rather below the left thalamus, the posterior limit of the cyst reaching to one and a half centimetres anterior to the posterior edge of the optic thalamus. The cyst was external to the thalamus, half a centimetre of white matter intervening between the cyst and the grey matter of the thalamus. Thus, posteriorly, the cyst was situated in and surrounded by the left peduncular expansion. Close to the posterior part of the corpus the cyst was only a quarter of a centimetre from the grey matter of one of the convolutions.

The cyst measured transversely one and a half centimetres, and its long measurement was about three centimetres.

The cavity was lined by loose connective tissue containing serosity, and the tissue around the cavity was tinged a light orange colour.

The cyst, in general terms, lay anteriorly in the corpus striatum, being situated in the lenticular nucleus; behind it lay outside, and rather below the anterior two-thirds of the thalamus, but the posterior portion of the whole left corpus was softened, and the thalamus was wasted.

The parts posterior to the lesion, namely, the crus, the pons, the medulla, the floor of the fourth ventricle, and the spinal cord and nerves, were carefully examined, but no evidence of wasting, or any other change, could be detected by the naked eye.

Dr. Ewart kindly took charge of the brain, and has made a careful microscopical examination which I append. He also has given me an account of the situation of the cyst and has kindly made the drawing showing its position. He says "the cyst lies in the posterior half or two-thirds of the lenticular nucleus, the cyst lying in front immediately without the inner capsule, but passing obliquely through the lenticular nucleus. After leaving the posterior part of the lenticular nucleus it involves the whole of the white matter lying outside and rather beneath the optic thalamus, and runs parallel with the left thalamus for about half an inch. A small portion of the grey matter of the optic thalamus adjacent to the cyst is destroyed."

I now give the drawings prepared by Dr. Ewart. The first represents a transverse section through the anterior part of the left corpus striatum, the second through the posterior portion, and the third through the optic thalamus.

"*Microscopic Appearances presented by Transverse Sections through the Structures surrounding the Anterior Third of Cyst.*—The most striking appearance presented by sections through the anterior end of cyst was the projection into its interior of fibres derived in part from the inner capsule (Fig. 1, C), but chiefly from the lenticular nucleus (Fig. 1, A). A few of the fibres extended in a straight line for 2—3 m.m. towards the centre, but the greater number formed an irregular network, supported by the empty blood-vessels, immediately within the cyst. In the meshes of this network were nerve-cells, blood-corpuscles and pigment masses. On the inner side many of the fibres were traceable to the inner capsule, some passing towards the surface, others towards the spinal-cord. At the lower and outer side the fibres were more delicate, and many of them from their connection with small round and oval nuclei were connective tissue-fibres, whilst others from their connection with large nerve-cells were evidently nerve-fibres. Other fibres passing from the peduncular expansion either entered to end in the cyst or passing along its outer side communicated with cells from which processes entered the cyst. The greater number of

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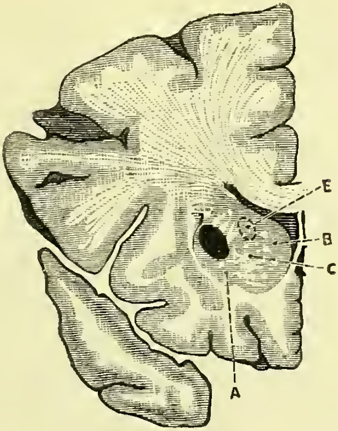

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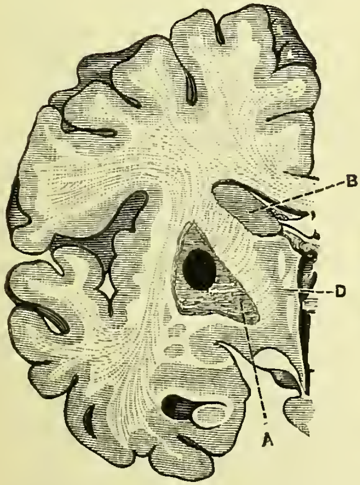

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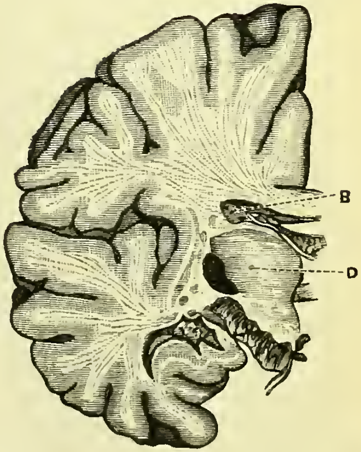

the fibres radiating from the cyst were lost sight of in the substance of the lenticular nucleus. Lying on and between the nerve- and connective tissue-fibres were many small round and oval nuclei, a few blood-corpuscles, and in several sections large oval (in transverse section round) sacs packed full of red blood-corpuscles. These sacs had probably resulted from the reflux of blood from the surrounding healthy substance into thin and unsupported veins. They were never found at the side of the cyst next the inner capsule. The arteries were empty and served as a supporting framework for connective tissue and nerve-fibres which had escaped the softening process; the nuclei in the walls of the vessels were very distinct but apparently healthy."

"To pass now to the ganglionic nerve-cells, and first those lying isolated or in groups amongst the fibres lining the cyst. A number of them looked quite healthy and had either long or short processes in connection with them, some of which passed to cells lying in the substance of the nucleus. From such cells in which the only visible change was a slightly granular condition of the nucleus to those of which scarcely anything but a coarsely granular nucleus was left, every possible gradation existed, the amount of degeneration being in ratio to the distance from the healthy structures. While the first appearance of atrophy seems to be a granular condition of the nucleus, the protoplasm around the nucleus seems first to undergo complete disintegration. Many of the cells adjacent to the cyst, and from which processes passed to its interior, had so atrophied that they would in all probability be functionally useless, but the cells external to these had large bright nuclei and nucleoli, and the processes passing to and from them were larger than usual. This apparent hypertrophy of the cells and fibres connected with them may be accounted for by supposing that besides their own work, they had done at least part of the work of the cells and fibres which had been destroyed."

"*Sections through the Middle Third of the Cyst* (Fig. 2) presented similar appearances, but on account of its lying deeper in the substance of the ganglion its relation to the inner capsule and the peduncular expansion was less apparent. The nerve-cells were similar to those already described, but less numerous, and the degenerative changes penetrated further into the centre of the ganglion. Numerous small bundles of nerve-fibres ended abruptly in the cyst and were surrounded with connective tissue-fibres and cells, blood-corpuscles, and pigment."

"*Sections through the Posterior Third of the Cyst* (Fig. 3) were especially interesting, for at this part the cyst had all but left the lenticular nucleus to involve the outer and lower surface of the optic thalamus and the fibres of the outer capsule lying between it and the claustrum."

"Numerous very delicate nerve-fibres extended into the cyst from the thalamus, and along with them, as before, connective tissue-fibres and blood-vessels. There were no widely dilated veins, but many isolated red blood-corpuscles. The nerve-cells in the irregular lining of the cyst as well as those in the substance of the thalamus were less distinct than in the other sections, and it was almost impossible to make out their relation to the healthy and partly-destroyed fibres. The fibres on the outer side of the cyst, however, were apparent enough, and the ruptured and degenerated fibres passing from it were traceable for some distance in both directions. From two to four millimetres of the grey substance of the thalamus round the cyst had lost its normal appearance, many granules were present, the fibres were indistinct, and the cells faintly granular."

"*Changes in the Caudate Nucleus.*—Near the inner capsule and parallel to the depression on the surface of the caudate nucleus already mentioned a tract of grey matter was found in which atrophic changes had evidently set in. The intercellular substance had partly disappeared, the cells almost obscured by free blood-corpuscles, and granules were lying near each other, and the fibres passing to and from them were evidently in an unhealthy state. The above changes only existed in the part of the caudate nucleus corresponding in extent to the anterior fourth of the cyst; their position is indicated at E, Fig. 1."

"The fibres and cells of the claustrum and of the other parts of the left hemisphere near the basal ganglia and on the surface were normal in appearance."

"No change could be detected in the spinal-cord, and there was only doubtful evidence of slight atrophic changes in a few of the fibres of the anterior pyramid in sections below the locus niger."

From the above it will be evident that a few of the motor fibres (inner capsule) passing between the lenticular and caudate nucleus and a greater number of the sensory fibres (outer capsule) passing from the filament were destroyed; that about one-sixth of the grey matter of the lenticular nucleus and a small portion of the lower and outer part of the optic thalamus was also completely destroyed, and a portion of what remained of these nuclei and of the caudate nucleus partly atrophied, and further that the fibres passing to and radiating from the destroyed grey matter would be rendered inactive.

The disease in this case therefore appears to have been limited to the optic thalamus, corpus striatum, and the parts just external. The whole of the corpus striatum was much damaged, both the intra- and extra-ventricular portions (caudate and lenticular nuclei). Beside much atrophy and slight degeneration of the intra-ventricular portion, about one-fifth of the lenticular ganglion was destroyed and was occupied by the anterior part of the cyst. A few of the fibres (inner capsule) passing between the nuclei of the corpus striatum were destroyed. The corpus striatum was the part which suffered most, but the left optic thalamus was also wasted, and a small portion of the lower and outer portion of this body was completely destroyed, whilst a considerable portion of the white matter external to the thalamus (sensory fibres of the outer capsule passing from the filament) was destroyed, and the part occupied by the posterior part of the cyst. It thus appears that the diagnosis made when the man first came under my care is (in the main) correct.

In the account published in the *Practitioner* (August, 1877), I said, "Dazzling before the eyes, dimness of sight, giddiness preceding loss of consciousness, and followed by loss of speech, and sensation and motion of the right side, point conclusively to the left hemisphere of the cerebrum as the seat of the disease. The giddiness indicates the mesencephalon, the loss of speech, the posterior part of the third frontal convolution, the loss of sensation, the thalamus opticus, and the loss of motion the corpus striatum, as the parts probably affected. As speech returned before sensation and sensation before voluntary motion, the main stress of the disease must have fallen on the corpus striatum and in a less degree on the thalamus. It is probable, I think, that the cause of the attack was an embolon set free from the diseased mitral valves blocking the middle cerebral artery."

It will conduce to the better understanding of this interesting case if I here introduce an account given me by Dr. Ewart of the probable course of the nerve fibres.

A considerable number, about one-fourth, of the fibres pass from the crus cerebri to the cortex without being in connection with the cells of the thalamus and corpus, some of the fibres passing outside these bodies.

The motor fibres running along the crusta radiate to all parts of the cerebrum to the frontal, parietal, occipital, and sphenoidal lobes, but especially to the frontal and parietal. One set of fibres passes directly to the occipital and temporal lobes, and another passes between the caudate and lenticular nuclei to the parietal and frontal lobes without any connection with the grey matter of the corpus. The remaining fibres of the crus pass into the grey matter of the corpus and terminate in the cells, and this part is in connection with the fibres which pass to the frontal and parietal lobes, and the apex of the temporal and to the insula. The olivary fasciculus from the anterior column, the fasciculi teretes from the lateral column, and fibres from the posterior pyramid, unite to form the tegmentum from which fibres pass to the corpora quadrigemena, corpora geniculata, and optic thalamus, and proceed from the thalamus to form part of the peduncular expansion and pass to the occipital parietal and tempero-sphenoidal convolutions and the insula. Other fibres undoubtedly pass directly to the cortex.

The consideration of this case naturally falls into two parts :—

1. The period of paralysis.
2. The period of athetosis.

It is interesting to observe that this man recovered completely from paralysis, both of motion and of sensation, although a large hole tunnelled through a most important part of his brain remained, and the tissue around it, especially that composing the left basic ganglia, continued considerably damaged. A not inconsiderable and a highly important part of the left cortex was therefore permanently cut off from the body and yet no paralysis remained. No doubt both in effusion of blood and in embolism much brain substance around the effused clot or around the tissue deprived of its blood, becomes affected by pressure, serous effusion, &c., and as the pressure is removed, the paralysis due to these causes subside. But in this case a serious lesion destroying a large extent of brain persisted, and yet the patient recovered completely from his paralysis. How is this to be explained ? The broken communication by means of the cyst between the cortex and the rest of the body could be re-established in two ways : by vicarious function, parts of the brain taking on function formerly performed by other portions of the brain ; or new channels for the passage of the nerve current arrested by the cyst might be formed around it.

This case does not enable me to say which of these views is correct, but I may suggest if the restoration of lost power was due to vicarious function, then that part of the cortex cut off by the cyst and no longer exercised should waste, but we could not distinctly detect any wasting of the convolutions.

I may point out that the lesion was situated in the usual seat in cases of hemianæsthesia and unilateral sweating, that is in the internal capsule outside and just below the thalamus opticus.

This case teaches us much regarding the seat and nature of the change in athetosis.

It is obvious that the cavity in the brain could not be the immediate cause of the incoordinated and involuntary movement of athetosis, for this breach of continuity of the brain could only cause paralysis. The athetosis therefore must be attributed to the damaged tissues in the neighbourhood of this cavity, and must in this case be due to the damage in the optic thalamus, or the corpus striatum, or both.

These are the structures in which one would naturally expect to find the lesions. Dr. Hammond in his original communication suggested the grey matter of the optic thalamus or the corpus striatum as the probable seat of disease.

As regards the nature of the disease, this case shows that it is due to atrophy and degeneration of the basal ganglia.

How does this atrophy and degeneration produce the movements occurring in athetosis? Dr. Gowers in his paper published in the *Medico-Chirurgical Transactions* for 1876, says:—"The slow irregular spasm which occurs on movement is clearly due in part to a diversion of the motor impulse along an unintended path, or its irradiation over a wider region than that to which it should have been confined." "The symptoms point clearly to damage to the grey matter of the brain, to local perverted nutrition of nerve-cells, in consequence of which they over-act either spontaneously or on the stimulus of the volitional impulse which is by their over-action perverted or irregularly distributed."

I beg to add the views I suggested in my previous account of this interesting case:—

The disease in this man does not imply destruction of function, there being no paralysis, but rather a perversion of function.

In this case we have the following four circumstances to consider:

1. There is, when awake, continuous evolution of force.
2. Incoordinate action.
3. Excessive evolution of force from normal stimuli of will, emotion, and reflex irritation.
4. This evolution of force is produced by stimuli which normally ought not at all to affect this part of the nervous system.

The most striking symptom is incoordinated action. Any stimulus originating either in the will or in the emotional centres no longer calls forth a definite and coordinated act, but on many occasions, and always, after a time, an utterly purposeless act. Yet it is something more than a mere incoordination; for assuming that for every complex combination of muscular contraction there is a coordinating centre, the disease does not consist merely of a destruction or weakening of some of these centres, for then we should get only irregular (incoordinated) action of the muscles set in motion by the voluntary act, but in addition to these many other muscles become powerfully affected; muscles even in the limb not set in motion by the will. Thus the stimulus, whether voluntary or emotional, does not run along definite channels, but radiates in all directions throughout the diseased area of grey matter, producing disorderly movements;

in other words, that force or condition of the nervous centres which restrains the discharge within certain definite areas of the grey matter is weakened, and, owing to this diminished resistance, the stimulus radiates into other parts, producing disorderly and widespread movements.

As in the case of allied spasmodic diseases, as chorea, strange to say, stimuli directed to the diseased nervous tissue not only produce disorderly movements, but impressions directed to a wholly different part find their way to the diseased grey matter, and radiating throughout it, produce disorderly movements. For instance, a voluntary stimulus intended to contract a part of the healthy arm or leg is partly or even wholly diverted to the diseased portion, and sets free in it a nervous discharge instead of in the part it was intended for. In this case emotional stimuli were more diverted by the diseased grey matter than volitional. We may assume that in this case, through some defect in resistance, impressions are not restricted to their proper channels; but at some point where the resistance fails the nervous force leaks out, finding its way to the diseased nervous structures connected with the right arm and leg.

Some eminent writers would, I believe, refer this evolution of force in the diseased nervous centre to higher nutrition reaching an explosive degree; and they would say that in Samson's case an ordinary volitional or emotional stimulus liberated an unusual amount of force, producing very powerful muscular contraction. It appears to me there are cogent objections to this view. We have seen that voluntary movement increases the incoordinated movement, and the longer the voluntary movement continues, the greater the incoordinated becomes, till at last incoordinated completely usurps coordinated movement. Now according to the foregoing theory, the voluntary movement should, so to speak, work off the higher nutrition, and keep it down below an explosive point, and the longer the persistence of the voluntary movement, the more the nutrition (potential force) is consumed, and the incoordinated should give way to coordinated action. On the supposition that the lesion has lessened and in some parts destroyed the resistance, the effect of voluntary and reflex movement is easy to explain. If depressed, then, by voluntary and reflex action it becomes still further depressed, and hence the impulse can more readily irradiate. The condition is comparable to that of the spinal cord of a frog to which a dose of strychnia, only just sufficient to produce tetanus, has been given. On irritating a limb we produce at first only a coordinated reflex act, but by keeping up the reflex contraction of the leg this at last becomes tetanic. The strychnia has depressed resistance, though not enough to permit the onset of tetanus till resistance itself is still more depressed by the functional activity of the cord. If it is said, How then do you explain the excessive evolution of force, and consequently very powerful muscular contraction ?—this I would explain by adducing a fact I have tried experimentally to prove, that the resistance not only localises, but restrains the amount of nervous discharge, and by weakening it we not only allow the irritation to irradiate, but also to set free an excessive amount of force.

Is any of the spasm in Samson's case due to the lesion which produced the loss of resistance ? Does the lesion act as an excitor causing a perpetual discharge of force, or is all the spasm due to volitional, emotional, and reflex stimuli ? In the early stage of the case, when the movements were violent during sleep, I suggest that the lesion acted as a stimulus, since it is evident that the movements could not be produced during sleep by the will or by the emotions. Latterly, and during the time he has been under our charge, the original lesion appears to play no part in the evolution of force, for the limbs are quiet during sleep. It is true, he tells us, that occasionally his arm or leg moves during sleep to wake him ; but after careful watching we have never seen this ; therefore it must occur but seldom, and I suggest that it is then due to the emotional excitement of a dream.

It seems to me that athetosis is allied in many diseases, with which at first sight it would appear to have little or no affinity. In athetosis, as we have seen, a change takes place in certain parts of the cerebrum, especially in the basal ganglia (optic thalamus and corpus striatum), whereby a stimulus is not restrained to its normal portion of grey matter, but " radiates " into other parts, so that instead of coordinated we get incoordinated muscular contraction. In addition, stimuli destined for another part of the brain are diverted as it were to the diseased area; and hence the diseased area becomes excited by stimuli which naturally would exert no action on it. In the case of many other diseases we meet with precisely the same unrestrained action, due to lessening or destruction of the "resistance" in certain portions of the cerebral nervous system, so that impressions radiate beyond their normal sphere. Wherever this irradiation or loss of "resistance" occurs, the change causing it, I venture to assume, is identical, whatever the kind of disease leading to it; being in the present case probably embolism. If this view holds good, it is obvious that athetosis becomes connected with many diseases manifesting symptoms widely different from those pertaining to athetosis itself. If the "loss of resistance," or in other words, the condition permitting "irradiation," is situated in parts connected with motor nerves, we get irregular incoordinated contraction of muscles; if with parts connected with sensory nerves, we get widespread pains. The persistence, remittence, or intermittence of the exciting cause will of course modify the symptoms. Thus if the exciting cause is permanent, then the muscular contraction or pain will be permanent; if remittent, the contraction or pain will be remittent; if intermittent, then also the contraction or pain will be intermittent.

Thus the name athetosis indicates the seat of the disease rather than its nature.

The constant slow movement due to the consecutive slow contraction and relaxation of different muscles can, I think, be explained in this wise: the loss of resistance being partial only, as soon as the evolution of force has reduced the potential force, the remaining resistance is adequate to restrain the irradiation, till nutrition has again restored and accumulated the potential force, when the weakened resistance is unable longer to restrain the evolution of force within normal limits. Contraction and relaxation of various muscles will then occur with consequent constant changes of the affected limb.

The similarity between athetosis and chorea is too evident to be dwelt on; so I will now attempt to show its affinity to diseases with which at first sight it would seem to have no relation. For example, it appears to me to be allied to stammering. In stammering, before the word can be uttered, there occurs, in mild cases, remittent and alternating spasm of the muscles surrounding the mouth; in more determined stammering almost all the muscles supplied by the seventh nerve are affected; in severe cases the neck muscles produce frequent spasmodical jerking of the head; whilst in very severe cases, in addition to all these muscular movements, there is frequent spasmodic heaving of the shoulders.

On analysing these movements, we find that the impulse, starting from the speech centre, instead of running at once in its proper channels, is diverted, and through loss of resistance radiates through the nucleus of the seventh, the irradiation being in mild cases slight, and involving that part only of the nucleus supplying the muscles surrounding the mouth. In more developed cases, however, irradiation takes place throughout the greater part of the nucleus of the seventh, and in still severer instances involves the grey matter in connection with nerves supplying some of the neck muscles, and reaches even the spinal accessory, causing spasmodic contraction of the trapezius and consequent heaving of the shoulders. As in athetosis, so in stammering, the muscles are not all simultaneously contracted, some being relaxed, whilst others are contracted. This state is not, of course, continuous like athetosis, for the simple reason that the exciting cause, speech, is itself intermittent; but I venture to think that the condition (the loss of resistance) permitting the irradiation is the same, only that it affects different parts, and stammering might therefore be called intermittent athetosis of the facial and other muscles.

I will give an illustrative example in the case of a man belonging to a family of stutterers, himself when young a notable stutterer. After a time the spasmodic movements of the face and of the head ceased, leaving in their stead a spasmodic shrugging of the right shoulder three or four times repeated before he could begin to speak, when the shoulder became quiet. Like ordinary stammering, the convulsion was much more marked, indeed was almost violent, when he tried to pronounce C and Z. Whilst the shrugging was going on he meanwhile was obviously striving strenuously to get out the word or letter. Here, the defect permitting irradiation, the loss of resistance, at first extensive, gradually grew less; but instead of disappearing entirely, as in most cases, a remnant remained, the impulse generated in the act of speech irradiating to the nucleus of the right spinal accessory nerve.

Athetosis is allied also to painful spasmodic affections, as neuralgia. But here the irradiation occurs in a portion of the grey matter associated with sensory nerves. Indeed, in watching over a case of athetosis we were struck with its similarity to a neuralgia. Thus we might compare it to a case of sciatica. In sciatica there is often some persistent pain, and in our case there was some sustained spasm. In sciatica, in addition to the characteristic persistent pain, other pains shoot now through one branch of the nerve, now dart through another. So with athetosis; in addition to the sustained contraction we get likewise now contraction of one set of muscles, and these relaxing, then contraction of another set.

It may be objected that in athetosis the irradiation is comparatively tardy, as shown by the slow continuous movement, whilst in stammering it is far quicker, and in neuralgia is of lightning-like rapidity: and that this difference must imply a difference in the nature of the change permitting the irradiation. To this I may answer that the rapidity of movement varies greatly in athetosis, as shown in the cases reported by Dr. Gowers; in other diseases the rapidity with which the nervous discharge travels varies greatly; thus the aura of epilepsy and the allied aura in other nervous affections, vary much in the rate of passage along the central nervous system, yet we do not therefore assume that the nature of the change in the nervous centres is different in kind.